WHY ARE SLOTHS SO SLOTHFUL?

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An alternative title to my presentation today could be "Are sloths myotonic?" They certainly have some characteristics which suggest it and it is my purpose to bring these to your attention.

You will recall that myotonia is an abnormality of skeletal muscle in which movement is slow in performance and delayed in cessation. It is more pronounced in limb than in bulbar muscles and is characterized by episodes of prolonged muscle contraction in which the limbs are immobilized by simultaneous tonic contraction of agonist and antagonist muscles.[1]

Patients who suffer with myotonia experience difficulty in initiating sudden actions and in releasing hand grip after grasping of an object.

One form of myotonia is myotonia congenita or Thomsen's disease, named after the Danish physician who suffered with it and who described its symptoms and hereditary characteristics in 1876.[2]

While myotonia congenita is rare, myotonia as one feature of myotonic dystrophy is not, and paramyotonia or the dramatic increase in myotonia which occurs when muscles are cooled is a common accompaniment of myotonic dystrophy.

Empirically it has been found that quinine and procaine amide provide relief from this disabling symptom.

There are two theories as to its causation, the first suggested by Denny-Brown and Nevin[3] that the disorder is caused by an abnormality in the nervous system and the second by an abnormality in the muscle itself as espoused by Landau.[4]

My interest in the sloth and the possibility that it has a variant form of myotonia was kindled during a sabbatical year that I spent at the Scripps Institution of Oceanography with Professor T. H. Bullock who is well known to all of you as a neuro-scientist and marine biologist.[5]

There are two genera of living sloths—Choloepus and Bradypus.[6] Both belong to the order Edentata, suborder Zenarthra along with armadillos and new world anteaters. This unique order has always been confined to the new world, fossil remains having been found in tar pits at Rancho La Brea (exhibited at the Los Angeles County Museum) as well as in caves

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throughout North American and Central and South America. Of interest is the fact that one of the earliest paleontologic studies of the giant ground sloth was that of Thomas Jefferson who read a paper before the American Philosophical Society in 1799 describing the results of his investigations of its fossil remains found in limestone caverns of western Virginia. His name is associated with the species of ground sloth widely found in Pleistocene fauna on the North American continent.[7]

The remarkably slow characteristics of these animals are apparent to all who have seen them (indeed one of the seven cardinal sins is sloth) but these primitive mammals whose habitat is the rain forests of Central and South America seldom have been studied. They spend their lives hanging inverted from limbs of trees grazing contentedly on cecropia leaves and sleeping 18–22 of every 24 hours.

Everyone who has studied the sloth has been impressed with the fact that they remain almost motionless for days at a time, with their slow movements, probably the slowest of any mammal, with their pitifully weak extensor muscles and with their powerful flexors which provide them with the unique capacity to support their weight with their hind limbs for minutes at a time at 90° to gravity.[8]

Britton concluded that the reason for the slow locomotion of the sloth was that it was hypothyroid.[9] Irving, Scholander and Grinnell confirmed that the animal was hypometabolic.[10] Goffart[11] and then Enger and Bullock[12] addressed themselves to the problem of why the animal remains immobile even in times of life threatening stress and considered two primary possibilities: a) that its inactivity might be due to adaptation to its environment and b) that the animal might have some response limiting factor which restricts its ability to react. To test the former possibility they exposed sloths to their natural enemies, they separated infants from mothers to bring out maternal instinct, and they dropped them to test righting reflexes. In all instances the animals remained docile and bradykinetic.

To test the second possibility, they measured central response times, synaptic transmission times, motor nerve conduction velocities, and neuromuscular junction delay. All were somewhat slow but the delay was not sufficient to account for the indolent actions of the sloth. Consequently, they measured the rate of development of tension in muscle at tetanizing frequencies and found that even though muscle action potentials were normal, contraction and relaxation times were remarkably slow. In all muscles the stimulus frequency at which tetany occurs is so low that it was concluded that the rate limiting mechanism was a combination of nervous system conduction and muscle mechanics.

I recalled the myotonic goats reported by Brown and Harvey[13], then embarked on a series of experiments to see if the sloth had any of the characteristics of myotonia. Six Choloepus and four Bradypus were imported to the Scripps Institution of Oceanography from Panama and were maintained there on a diet of cecropia leaves. Because of their slow movements, it was possible to perform neurological examinations on unsedated wild animals. In both species the elicitation of muscle stretch reflex was always associated with an enormous spread to other muscle groups. For example, a gentle tap on the tendon of the biceps brachii or pectoralis major produced contraction of the flexors of the entire limb on the same and usually the opposite side as well. Light percussion of the manubrium sterni caused contraction of the flexors of all four limbs simultaneously.

Spontaneous movements and stretch reflexes were slowed when rectal temperature was reduced from a normal of 33 to 30° C and muscle temperature from 28 to 21° C by immersion in 21° sea water. At no time, however, did these animals develop contracture. When cooled to these temperatures, tapping of the muscle belly of the biceps, the tongue, or the flexor digitorium produced a sustained local contraction with slow relaxation time suggesting myotonia. In contrast, elevation of the body temperature by heating the animals to a rectal temperature of 38° and muscle temperature of 36° did not speed muscular response particularly.

Electromyography of muscle activity did not show any of the insertional potentials of "dive bomber" sounds commonly associated with myotonia.

Pupillary responses also show a very slow response to light. Mydriasis required several hours of dark adaptation and on reexposure to light the pupils slowly became smaller taking about five minutes for full constriction. They seemed unusually sensitive to the use of mecholyl and surprisingly enough light shown into one eye cause slow constriction of that pupil alone.

Of the two species, Bradypus showed the more exaggerated or slower responses.

In anesthetized animals, the median nerve was isolated in the antecubital space and the tendon of the flexor digitorum profundus was attached to a force transducer for isometric recording. The output of the transducer was recorded on a strip chart and the brachial artery was cannulated for the installation of drugs and for sampling of blood. The mechanogram was studied for possible effects of change of muscle temperature, frequency of nerve stimulation and various pharmacological agents. Skeletal muscle biopsies were obtained for histological, histochemical, and biochemical analysis.

A summary of these studies is:

1) Despite supramaximal stimuli to the nerve, muscle contraction facilitated for the first several contractions.

2) Relaxation at the end of a train of stimuli was somewhat slow as compared to normal skeletal muscle, but rapid as compared to myotonic muscle.
3) Quinine, procaine amide, calcium gluconate did not alter this response.
4) Cutting the nerve proximal to the point of stimulation did not alter the response characteristics.
5) Direct muscle stimulation did not alter the response characteristics.
6) The muscles are histologically normal but they contain only red muscle in contrast to most mammals which have a mixture of red and white muscle.[14]
7) The EDTA ATPase activity is the lowest of any mammal ever tested by Dr. Frank Sreter[15] who kindly analyzed our muscle specimens for us.

The result of these experiments is that we cannot verify our initial suspicion that the sloth might be myotonic. Electromyography did not demonstrate the myotonic response nor did procaine amide or quinine have an effect. However, percussion of muscles did produce an immediate contraction and mounding the muscle at the site of the blow which persisted for an appreciable length of time before slowly relaxing and the muscle was quite sensitive to cold. These findings would suggest that further studies of this unique animal would be in order.

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DISCUSSION

DR. HUGH MONTGOMERY (Philadelphia): What makes it possible for these animals to live in the presence of predators? Are they no good to eat or do they have some defense characteristics that we don't know?

DR. TOOLE: You have brought up a fascinating aspect of this animal's adaptation. How does this primitive animal, obviously unable to compete in aggressive jungle society, manage to survive? Either he is not delicious or the other animals can't find him. I've never tried a sloth so I can't tell you whether he's delicious or not! But there is a group who believe that because it moves so slowly, other animals accustomed to the darting figures of more fleet-footed animals miss this animal in the jungle. Furthermore, it hangs high and way out at the ends of limbs in the canopy of the jungle and as a consequence is not easily accessible.

DR. LEWIS DEXTER (Boston): Two points. You mentioned very briefly that they have very weak extensor muscles. Do they also have a weakness of all muscles or is it just the extensors? Secondly, you said that they were hypothyroid. As a simple clinician, I wondered if you'd given them any thyroid to brisk them up?

DR. TOOLE: First, they have very strong flexor muscles. They are able to hang at 90° to gravity with their hind limbs for minutes at a time, a feat that few other animals can do. Secondly, to say that they are hypothyroid by our standards may not mean that they are hypothyroid by sloth standards. Their serum cholesterols are high. Their basal metabolic rate is presumably about −50%. Their PBI is low as I've mentioned. I have not given them anything to speed them up but Britton who studied them did. He gave them desiccated thyroid, epinephrine, benzedrine and a variety of other agents, and nothing happened to their locomotion.

DR. BELTON A. BURROWS (Boston): Have you obtained any index of thyroxin binding properties of serum proteins because a low PBI could be associated with a perfectly normal thyroxin turnover?

DR. TOOLE: We did not do any partitions. We did an electrophoretic spread on these animals but I didn't do bound and unbound calcium, or thyroid or thyroxin, and so I can't answer that question.

DR. RICHARD S. ROSS (Baltimore): Do you have any information about how the heart works? For example, what is the heart rate of the sloth and what does its electrocardiogram show?

DR. TOOLE: The heart rate is very slow. It, however, is an unusual heart. Dr. Peter Poole at San Diego who studied the ability of the heart to contract was astonished, as was I, that animals thought to be dead continued to have heart beat when it was removed from the body much as the turtle heart might. Even when put in a calcium-free perfusion for twenty-four hours, the heart papillary muscle continued to contract and evidently was almost indestructible.

DR. ROBERT AUSTRIAN (Philadelphia): When we were in Burma, we had a pet that was a slow loris. I wonder if there is any similarity between this animal and the sloth, and whether you would like to study this on your next sabbatical.

DR. TOOLE: I certainly want another sabbatical, and I would love to study the loris. I don't know anything about it.